UNITED STATES PATENT AND TRADEMARK OFFICE

UNITED STATES DEPARTMENT OF COMMERCE
United States Patent and Trademark Office
Address: COMMISSIONER FOR PATENTS
P.O. Box 1450
Alexandria, Virginia 22313-1450
www.uspto.gov

APPLICATION NO.	FILING DATE	FIRST NAMED INVENTOR	ATTORNEY DOCKET NO.	CONFIRMATION NO.
10/565,591	10/06/2006	David K.R. Karaolis	KARAOLIS1A	2282

1444 7590 07/09/2009

BROWDY AND NEIMARK, P.L.L.C.
624 NINTH STREET, NW
SUITE 300
WASHINGTON, DC 20001-5303

EXAMINER	
ARCHIE, NINA	
ART UNIT	PAPER NUMBER
1645	
MAIL DATE	DELIVERY MODE
07/09/2009	PAPER

Please find below and/or attached an Office communication concerning this application or proceeding.

The time period for reply, if any, is set in the attached communication.

Office Action Summary	Application No.	Applicant(s)	
	10/565,591	KARAOLIS, DAVID K.R.	
	Examiner	Art Unit	
	Nina A. Archie	1645	

-- The MAILING DATE of this communication appears on the cover sheet with the correspondence address --

Period for Reply

A SHORTENED STATUTORY PERIOD FOR REPLY IS SET TO EXPIRE *3* MONTH(S) OR THIRTY (30) DAYS, WHICHEVER IS LONGER, FROM THE MAILING DATE OF THIS COMMUNICATION.

- Extensions of time may be available under the provisions of 37 CFR 1.136(a). In no event, however, may a reply be timely filed after SIX (6) MONTHS from the mailing date of this communication.
- If NO period for reply is specified above, the maximum statutory period will apply and will expire SIX (6) MONTHS from the mailing date of this communication.
- Failure to reply within the set or extended period for reply will, by statute, cause the application to become ABANDONED (35 U.S.C. § 133). Any reply received by the Office later than three months after the mailing date of this communication, even if timely filed, may reduce any earned patent term adjustment. See 37 CFR 1.704(b).

Status

1)☒ Responsive to communication(s) filed on *15 June 2009*.

2a)☒ This action is **FINAL**. 2b)☐ This action is non-final.

3)☐ Since this application is in condition for allowance except for formal matters, prosecution as to the merits is closed in accordance with the practice under *Ex parte Quayle*, 1935 C.D. 11, 453 O.G. 213.

Disposition of Claims

4)☒ Claim(s) *1-7,9-21 and 28-30* is/are pending in the application.

4a) Of the above claim(s) _____ is/are withdrawn from consideration.

5)☐ Claim(s) _____ is/are allowed.

6)☒ Claim(s) *1-7,9-21 and 28-30* is/are rejected.

7)☐ Claim(s) _____ is/are objected to.

8)☐ Claim(s) _____ are subject to restriction and/or election requirement.

Application Papers

9)☐ The specification is objected to by the Examiner.

10)☐ The drawing(s) filed on _____ is/are: a)☐ accepted or b)☐ objected to by the Examiner.

Applicant may not request that any objection to the drawing(s) be held in abeyance. See 37 CFR 1.85(a).

Replacement drawing sheet(s) including the correction is required if the drawing(s) is objected to. See 37 CFR 1.121(d).

11)☐ The oath or declaration is objected to by the Examiner. Note the attached Office Action or form PTO-152.

Priority under 35 U.S.C. § 119

12)☐ Acknowledgment is made of a claim for foreign priority under 35 U.S.C. § 119(a)-(d) or (f).

a)☐ All b)☐ Some * c)☐ None of:

1.☐ Certified copies of the priority documents have been received.

2.☐ Certified copies of the priority documents have been received in Application No. _____.

3.☐ Copies of the certified copies of the priority documents have been received in this National Stage application from the International Bureau (PCT Rule 17.2(a)).

* See the attached detailed Office action for a list of the certified copies not received.

Attachment(s)

1) ☒ Notice of References Cited (PTO-892)
2) ☐ Notice of Draftsperson's Patent Drawing Review (PTO-948)
3) ☒ Information Disclosure Statement(s) (PTO/SB/08) Paper No(s)/Mail Date *6/15/2009*.
4) ☐ Interview Summary (PTO-413) Paper No(s)/Mail Date. _____ .
5) ☐ Notice of Informal Patent Application
6) ☐ Other: _____.

DETAILED ACTION

Continued Examination Under 37 CFR 1.114

1. A request for continued examination under 37 CFR 1.114, including the fee set forth in 37 CFR 1.17(e), was filed in this application after final rejection. Since this application is eligible for continued examination under 37 CFR 1.114, and the fee set forth in 37 CFR 1.17(e) has been timely paid, the finality of the previous Office action has been withdrawn pursuant to 37 CFR 1.114. Applicant's submission filed on June 15, 2009 has been entered.

2. Claims 1-7, 9-12, 13-21, and 28-30 are pending and under examination.

Information Disclosure Statement

3. The information disclosure statement filed on 6/15/09 has been considered. Initialed copies are enclosed.

Claim Rejections Maintained

35 USC § 112

The following is a quotation of the first paragraph of 35 U.S.C. 112:

> The specification shall contain a written description of the invention, and of the manner and process of making and using it, in such full, clear, concise, and exact terms as to enable any person skilled in the art to which it pertains, or with which it is most nearly connected, to make and use the same and shall set forth the best mode contemplated by the inventor of carrying out his invention.

Enablement

4. The rejections of claims 1-5, 10-16, and 28-30 under 35 U.S.C. 112, first paragraph failing to comply with the enablement requirement are maintained for the reason set forth in the previous office action. The claim(s) contain subject matter, which was not described in the specification in such a way as to enable one skilled in the art to which it pertains, or with which it is most nearly connected, to make and/or use the invention.

As outlined previously: the specification, while being enabling for a method for attenuating the virulence of a microbial pathogen from *S. aureus or* for inhibiting or reducing colonization by a microbial pathogen from S. aureus in a patient in need thereof, comprising administering to the patient in need an effective amount of c-di-GMP, cGMP and 5'-GMP to attenuate the virulence of, or to inhibit or reduce colonization by, the microbial pathogen, does not reasonably provide enablement for any method for attenuating the virulence of any microbial pathogen or for inhibiting or reducing colonization by any microbial pathogen in a patient in need thereof, comprising administering to the patient in need an effective amount of c-di--GMP or a cyclic dinucleotide to attenuate the virulence of, or to inhibit or reduce colonization by, the microbial pathogen. The specification does not enable any person skilled in the art to which it pertains, or with which it is most nearly connected, to make and use the invention commensurate in scope with the claimed invention.

Nature of the invention. The claims are drawn to any method for attenuating the virulence of a microbial pathogen or for inhibiting or reducing colonization by a microbial pathogen in a patient in need thereof, comprising administering to the patient in need an effective amount of c-di-GMP or a cyclic dinucleotide to attenuate the virulence of, or to inhibit or reduce colonization by, the microbial pathogen.

The breadth of the claims. The method claim is very broad and the product, a cyclic dinucleotides used to administer to a patient is directed to any microbial pathogen. Furthermore the claims are drawn to any method for attenuating the virulence of a microbial pathogen or for inhibiting or reducing colonization of a microbial pathogen in a patient in need thereof, comprising administering to the patient in need an effective amount of any cyclic dinucleotide to attenuate the virulence of, or to inhibit or reduce colonization by, the microbial pathogen. Therefore it is hard for one skilled in the art to determine if any cyclic dinucleotide can be used to attenuate the virulence, inhibit or reduce the colonization or any microbial pathogen in a patient. Since the specification fails to provide particular guidance for any method for attenuating the virulence of a microbial pathogen or for inhibiting or reducing colonization of a microbial pathogen in a patient in need thereof, comprising administering to the patient in need an effective

amount of any type of a cyclic dinucleotide to attenuate the virulence of, or to inhibit or reduce colonization by, the microbial pathogen, it would require undue experimentation to practice the invention over the broad scope as presently claimed.

Guidance in the specification/Working Example. The specification discloses in Example 3 (see pp. 49-67), various examples, such as the effect of c-di-GMP on S. aureus biofilm formation (see 00101), the effects of c-di-GMP on S. aureus pre-formed biofilms (00102), c-di-GMP treatment the prevents cell to cell interaction (see 00111), c-di-GMP inhibiting biofilm formation in human and bovine S. aureus (see 00113), the effects of cGMP and 5'GMP on biofilm formation (see 00116), the effect of c-di-GMP treatment on S. aureus pre-formed biofilms (see 00117), and lastly safety and toxicity tests disclosing the treatment of c-di-GMP on mice that indicates to the inventor that c-di-GMP is relatively safe and not toxic (see 00119-00120). There is no showing in the specification that cyclic dinucleotides can be administered to a patient to attenuate the virulence of a microbial pathogen or for inhibiting or reducing colonization by a microbial pathogen. Although the specification gives several examples of a method for inhibiting microbial colonization and pre-formed microbial biofilm by disclosing various examples, such as in vitro studies of the effects c-di-GMP or any cyclic dinucleotides species there of on pre-formed microbial biofilm or biofilm formation and c-di-GMP treatment that prevents cell to cell interaction (see Example 3), the specification fails to show a method comprising administering to the patient in need an effective amount of c-di-GMP or any cyclic dinucleotide to attenuate the virulence of, or to inhibit or reduce colonization by, the microbial pathogen. Furthermore although the specification discloses orally administering c-di-GMP to mice that indicates to the inventor that c-di-GMP is relatively safe and not toxic only contemplates the claimed invention (see 00119-00120). The specification does not give any working example (i.e. challenged mice models or passive immunization approaches). Therefore the specification fails to describe any method for attenuating the virulence of any microbial pathogen or for inhibiting or reducing colonization by any microbial pathogen in a patient in need thereof.

The state of the prior art. The state of the art is unpredictable with regard to administering cyclic dinucleotides to attenuate the virulence of a microbial pathogen or for inhibiting or reducing colonization in a patient. The state of the art questions the correlation between in vivo and in vitro models for treatment of bacterial/microbial pathogens. For example, Parsek et al proposed four basic criteria to define biofilm-associated infections: (i) Bacterial cell adherence to or association with a surface, (ii) in vivo observation of bacterial cell clusters, (iii) a localized infection pattern, and (iv) increased resistance to antibiotic treatment in the host compared to resistance of genetically equivalent planktonic bacteria. A role for bacterial biofilms in pathogenesis is well established for a number of infections and opportunistic pathogens; for many other infections a link between biofilms and disease has been proposed, but the evidence remains less clear (see Parsek et al 2003. Bacterial biofilms: an emerging link to disease pathogenesis. Annu. Rev. Microbiol. 57:677-701 in its entirety). The state of the art indicate that Reisner et al teach the understanding of *Escherichia coli* biofilm formation in vitro is based on studies of laboratory K-12 strains grown in standard media. The data demonstrate that prevalence and expression of three factors known to strongly promote biofilm formation in *E. coli* K-12 (F-like conjugative pili, aggregative adherence fimbriae, and curli) cannot adequately account for the increased biofilm formation of nondomesticated *E. coli* isolates in vitro. Reisner et al discuss the complexity of genetic and environmental effectors of the biofilm phenotype within the species *E. coli*. Reisner et al teach the results found were a poor correlation between biofilm formation in different media, suggesting that *E. coli* isolates respond very differently to the changing growth and environmental conditions and that this finding emphasizes the relevance and difficulty involved in selecting proper conditions for in vitro biofilm studies which attempt to mirror natural environments in vivo. Reisner et al teach that based the results, in vitro biofilm phenotypes cannot be correlated with the expected virulence phenotypes of the *E. coli* isolates in vivo. Reisner et al further teach that a tremendous impact of environmental conditions highlights the need to develop better biofilm model systems to approximate in vivo situations. Furthermore careful adjustment of the medium composition is an important first step. Incorporation of more adequate surfaces in the experimental design appears to be an additional measure, e.g., by studying biofilm formation directly on eukaryotic cells. However, given that multiple species are present in most environments, we also need to establish models that enable monitoring of

possible antagonistic or synergistic interactions between community members (see Reisner et al 2006 Journal of Bacteriology Vol. 188 No. 10 pgs. 3572-3581 see abstract, pg. 3572 column 1 and pg. 3580). Furthermore the art indicates that device related infections are difficult to treat with antibiotics alone and that the minimum inhibitory concentrations (MICs) are not predictive for the therapeutic outcome in either the in vitro or in vivo model. For example the treatment of device related infections between the efficacy of antibiotics and the of drug levels of MICs is poor (see abstract and pg. 1138). Furthermore, the art indicates that the clinical relevance of susceptibility testing has always been questioned because of the difficulty of correlating in vitro susceptibility testing with in vivo clinical effectiveness and that there have always been host/pathogen factors that influence the clinical outcome that cannot be predicted by the results of susceptibility testing (see Stratton 2006 Med. Clin North Am Vol. 6 pgs. 1077-1088 see abstract). The state of the art teach that c-di-GMP is a novel naturally occurring nucleotide identified in prokaryotic systems and has found to be active in eukaryotic systems (see Steinberger et al 1999 FEBS LETTERS Vol. 444 pgs. 125-129 specifically pg. 125). Additionally Bowie et al (Science, 1990, 247:1306-1310) teach that an amino acid sequence encodes a message that determines the shape and function of a protein and that it is the ability of these proteins to fold into unique three-dimensional structures that allows them to function, carry out the instructions of the genome and form immunoepitopes. Bowie et al. further teach that the problem of predicting protein structure from sequence data and in turn utilizing predicted structural determinations to ascertain functional aspects of the protein is extremely complex. (column 1, page 1306). Bowie et al further teach that while it is known that many amino acid substitutions are possible in any given protein, the position within the protein's sequence where such amino acid substitutions can be made with a reasonable expectation of maintaining function are limited. Certain positions in the sequence are critical to the three dimensional structure/function relationship and these regions can tolerate only conservative substitutions or no substitutions (column 2, page 1306). Therefore the art questions whether any type of cyclic dinucleotides would have the same effect on the method as claimed.

Furthermore the art has not shown any method of administering any type of cyclic dinucleotides to attenuate the virulence of any microbial pathogen or for inhibiting or reducing colonization in a patient. The art questions the correlation between an in vivo and an in vitro

model. Therefore, given the lack of success in the art. For the reasons set forth supra, the state of the art is unpredictable in regards to administering any cyclic dinucleotide to attenuate the virulence of any microbial pathogen or for inhibiting or reducing colonization in a patient.

In conclusion, the claimed inventions are not enabled for any method for attenuating the virulence of any microbial pathogen or for inhibiting or reducing colonization by any microbial pathogen in a patient in need thereof, comprising administering to the patient in need an effective amount of c-di--GMP or a cyclic dinucleotide to attenuate the virulence of, or to inhibit or reduce colonization by, the microbial pathogen. The state of the art indicates that the clinical relevance of susceptibility testing has always been questioned because of the difficulty of correlating in vitro susceptibility testing with in vivo clinical effectiveness and that there have always been host/pathogen factors that influence the clinical outcome that cannot be predicted by the results of susceptibility testing (see Stratton 2006 Med. Clin North Am Vol. 6 pgs. 1077-1088 see abstract). The art has not shown any method of administering c-di--GMP or any cyclic dinucleotide to attenuate the virulence of any microbial pathogen or for inhibiting or reducing colonization in a patient. Furthermore, the art questions the correlation between an in vivo and an in vitro model. For the reasons set forth supra, the state of the art is unpredictable. There is also a lack of working examples. Although the specification discloses orally administering c-di-GMP to mice that indicates to the inventor that c-di-GMP is relatively safe and not toxic only contemplates the claimed invention. As a result, for the reasons discussed above, it would require undue experimentation for one skilled in the art to use the claimed methods.

Enablement

5. The rejection of claims 17-21 under 35 U.S.C. 112, first paragraph, as failing to comply with the enablement requirement are maintained for the reason set forth in the previous office action. The claim(s) contain subject matter, which was not described in the specification in such a way as to enable one skilled in the art to which it pertains, or with which it is most nearly connected, to make and/or use the invention.

As outlined previously, the specification, while being enabling for a method for inhibiting *Staphylococcus aureus (S. areus)* colonization and S. areus biofilm formation or for reducing S.

areus colonization and pre-formed *S. areus* microbial biofilm on a solid surface, comprising exposing the solid surface to an effective amount of c-di-GMP or a cyclic dinucleotide to inhibit S. areus colonization and S. areus biofilm formation or to reduce microbial colonization and pre-formed biofilm on said solid surface, does not reasonably provide enablement for any method for inhibiting bacterial colonization and biofilm formation or for reducing colonization and pre-formed bacterial biofilm on a solid surface, comprising exposing the solid surface to an effective amount of c-di-GMP or a cyclic dinucleotide to inhibit bacterial colonization and biofilm formation or to reduce bacterial colonization and pre-formed biofilm on said solid surface. The specification does not enable any person skilled in the art to which it pertains, or with which it is most nearly connected, to make and use the invention commensurate in scope with the claimed invention.

Nature of the invention. The claims are drawn to for any method for attenuating the virulence of a microbial pathogen or for inhibiting or reducing colonization by a microbial pathogen in a patient in need thereof, comprising administering to the patient in need an effective amount of c-di-GMP or a cyclic dinucleotide to attenuate the virulence of, or to inhibit or reduce colonization by, the microbial pathogen.

The breadth of the claims. The method claim is very broad and the product, a c-di-GMP or any cyclic dinucleotide used in the method as set for supra to any type of microbial colonization or any type biofilm formation. Furthermore the claims are drawn to any method for inhibiting any type microbial colonization and any type of biofilm formation or for reducing any type of colonization and pre-formed any type of microbial biofilm on a solid surface, comprising exposing the solid surface to an effective amount of a c-di-GMP or any cyclic dinucleotide to inhibit any type of microbial colonization and any biofilm formation or to reduce any microbial colonization and any pre-formed biofilm on said solid surface. Therefore it is hard for one skilled in the art to determine if c-di-GMP or any cyclic dinucleotide can be used in the method as set forth supra. The quantity of experimentation required to practice the invention as claimed would require studies of c-di-GMP or any cyclic dinucleotide to attenuate the virulence of, or to inhibit or reduce colonization of all types of microbial pathogens. Since the specification fails to provide

particular guidance for the method as set forth supra, it would require undue experimentation to practice the invention over the broad scope as presently claimed.

Guidance in the specification/Working Examples. The specification discloses in Example 3 (see pp. 49-67), various examples, such as the effect of c-di-GMP on S. aureus biofilm formation (see 00101), the effects of c-di-GMP on S. aureus pre-formed biofilms (00102), c-di-GMP treatment the prevents cell to cell interaction (see 00111), c-di-GMP inhibiting biofilm formation in human and bovine S. aureus (see 00113), the effects of cGMP and 5'GMP on biofilm formation (see 00116), the effect of c-di-GMP treatment on S. aureus pre-formed biofilms (see 00117), and lastly safety and toxicity tests disclosing the treatment of c-di-GMP on mice that indicates to the inventor that c-di-GMP is relatively safe and not toxic (see 00119-00120). The specification gives several examples of S. aureus bacteria in method for inhibiting microbial colonization and pre-formed microbial biofilm by disclosing various examples, such as in vitro studies of the effects c-di-GMP or any cyclic dinucleotide on pre-formed microbial biofilm or biofilm formation and c-di-GMP treatment that prevents cell to cell interaction (see Example 3. Furthermore Example 4 discloses extracellular c-di-GMP increases. Furthermore although the specification discloses orally administering c-di-GMP to mice that indicates to the inventor that c-di-GMP is relatively safe and not toxic only contemplates the claimed invention (see 00119-00120). Therefore the specification fails to describe any method for attenuating the virulence of a microbial pathogen or for inhibiting or reducing colonization by a microbial pathogen in a patient in need thereof, comprising administering to the patient in need an effective amount of c-di-GMP or a cyclic dinucleotide to attenuate the virulence of, or to inhibit or reduce colonization by, the microbial pathogen.

The state of the prior art. The state of the art is unpredictable with regard to c-di-GMP and inhibiting or reducing colonization and biofilm formation in microbial pathogens. state of the art teach that c-di-GMP is a novel naturally occurring nucleotide identified in prokaryotic systems and has found to be active in eukaryotic systems (see Steinberger et al 1999 FEBS LETTERS Vol. 444 pgs. 125-129 specifically pg. 125). Parsek et al proposed four basic criteria to define biofilm-associated infections: (i) Bacterial cell adherence to or association with a surface, (ii) in

vivo observation of bacterial cell clusters, (iii) a localized infection pattern, and (iv) increased resistance to antibiotic treatment in the host compared to resistance of genetically equivalent planktonic bacteria. A role for bacterial biofilms in pathogenesis is well established for a number of infections and opportunistic pathogens; for many other infections a link between biofilms and disease has been proposed, but the evidence remains less clear (see Parsek et al 2003. Bacterial biofilms: an emerging link to disease pathogenesis. Annu. Rev. Microbiol. 57:677-701 in its entirety). The state of the art indicate that Reisner et al teach the understanding of *Escherichia coli* biofilm formation in vitro is based on studies of laboratory K-12 strains grown in standard media. The data demonstrate that prevalence and expression of three factors known to strongly promote biofilm formation in *E. coli* K-12 (F-like conjugative pili, aggregative adherence fimbriae, and curli) cannot adequately account for the increased biofilm formation of nondomesticated *E. coli* isolates in vitro. Reisner et al discuss the complexity of genetic and environmental effectors of the biofilm phenotype within the species *E. coli*. Reisner et al teach the results found were a poor correlation between biofilm formation in different media, suggesting that *E. coli* isolates respond very differently to the changing growth and environmental conditions and that this finding emphasizes the relevance and difficulty involved in selecting proper conditions for in vitro biofilm studies which attempt to mirror natural environments in vivo. Reisner et al teach that based the results, in vitro biofilm phenotypes cannot be correlated with the expected virulence phenotypes of the *E. coli* isolates in vivo. Reisner et al further teach that a tremendous impact of environmental conditions highlights the need to develop better biofilm model systems to approximate in vivo situations. Furthermore careful adjustment of the medium composition is an important first step. Incorporation of more adequate surfaces in the experimental design appears to be an additional measure, e.g., by studying biofilm formation directly on eukaryotic cells. However, given that multiple species are present in most environments, we also need to establish models that enable monitoring of possible antagonistic or synergistic interactions between community members (see Reisner et al 2006 Journal of Bacteriology Vol. 188 No. 10 pgs. 3572-3581 see abstract, pg. 3572 column 1 and pg. 3580). Furthermore the art indicates that device related infections are difficult to treat with antibiotics alone and that the minimum inhibitory concentrations (MICs) are not predictive for the therapeutic outcome in either the in vitro or in vivo model. For example the treatment of

device related infections between the efficacy of antibiotics and the of drug levels of MICs is poor (see abstract and pg. 1138). Furthermore, the art indicates that the clinical relevance of susceptibility testing has always been questioned because of the difficulty of correlating in vitro susceptibility testing with in vivo clinical effectiveness and that there have always been host/pathogen factors that influence the clinical outcome that cannot be predicted by the results of susceptibility testing (see Stratton 2006 Med. Clin North Am Vol. 6 pgs. 1077-1088 see abstract).

The art questions biofilm model systems and the factors that have to be considers as set forth supra. Therefore, given the lack of success in the art the state of the art is unpredictable with regard to c-di-GMP and inhibiting or reducing colonization and biofilm formation in microbial pathogens.

In conclusion, the claimed invention is not enabled for any method for inhibiting bacterial colonization and biofilm formation or for reducing colonization and pre-formed bacterial biofilm on a solid surface, comprising exposing the solid surface to an effective amount of c-di-GMP or a cyclic dinucleotide to inhibit bacterial colonization and biofilm formation or to reduce bacterial colonization and pre-formed biofilm on said solid surface. The state of the art indicates that the clinical relevance of susceptibility testing has always been questioned because of the difficulty of correlating in vitro susceptibility testing with in vivo clinical effectiveness and that there have always been host/pathogen factors that influence the clinical outcome that cannot be predicted by the results of susceptibility testing (see Stratton 2006 Med. Clin North Am Vol. 6 pgs. 1077-1088 see abstract). The art questions biofilm model systems and the factors that have to be considers as set forth supra. Therefore, given the lack of success in the art the state of the art is unpredictable with regard to c-di-GMP and inhibiting or reducing colonization and biofilm formation in microbial pathogens. As a result, for the reasons discussed above, it would require undue experimentation for one skilled in the art to use the claimed methods.

Citation of Relevant Art

6. The prior art made of record and not relied upon is considered pertinent to applicant's disclosure.

Ross et al. Biol. Chem. 265 No. 31 (1990) 18933-18943 teaches the effects of c-di-GMP and other cyclic dinucleotides on cellulose synthetase.

Conclusion

7. No claims are allowed.

All claims are drawn to the same invention claimed in the application prior to the entry of the submission under 37 CFR 1.114 and could have been finally rejected on the grounds and art of record in the next Office action if they had been entered in the application prior to entry under 37 CFR 1.114. Accordingly, **THIS ACTION IS MADE FINAL** even though it is a first action after the filing of a request for continued examination and the submission under 37 CFR 1.114. See MPEP § 706.07(b). Applicant is reminded of the extension of time policy as set forth in 37 CFR 1.136(a).

A shortened statutory period for reply to this final action is set to expire THREE MONTHS from the mailing date of this action. In the event a first reply is filed within TWO MONTHS of the mailing date of this final action and the advisory action is not mailed until after the end of the THREE-MONTH shortened statutory period, then the shortened statutory period will expire on the date the advisory action is mailed, and any extension fee pursuant to 37 CFR 1.136(a) will be calculated from the mailing date of the advisory action. In no event, however, will the statutory period for reply expire later than SIX MONTHS from the mailing date of this final action.

Any inquiry concerning this communication or earlier communications from the examiner should be directed to Nina A. Archie whose telephone number is 571-272-9938. The examiner can normally be reached on Monday-Friday 8:30-5:00p.m.

If attempts to reach the examiner by telephone are unsuccessful, the examiner supervisor, Robert Mondesi can be reached on 571-272-0956. The fax phone number for the organization where this application or proceeding is assigned is 571-273-8300.

Information regarding the status of an application may be obtained from the Patent Application Information Retrieval (PAIR) system. Status information for published applications may be obtained from either Private PAIR or Public PAIR. Status information for unpublished

applications is available through Private PAIR only. For more information about the PAIR system, see http://pair-direct.uspto.gov. Should you have questions on access to the Private PAIR system, contact the Electronic Business Center (EBC) at 866-217-9197 (toll-free). If you would like assistance from a USPTO Customer Service Representative or access to the automated information system, call 800-786-9199 (IN USA OR CANADA) or 571-272-1000.

Nina A Archie
Examiner
GAU 1645
REM 3B31

/Robert A. Zeman/

for Nina Archie, Examiner of Art Unit 1645